CALIFORNIA
AND
WESTERN MEDICINE

VOLUME XXXII JANUARY, 1930 No. 1

INTESTINAL OBSTRUCTION*

SOME MEDICAL ASPECTS

By V. R. MASON, M. D.
Los Angeles

DISCUSSION *by Charles D. Lockwood, M. D., Pasadena; William J. Kerr, M. D., San Francisco; Lovell Langstroth, M. D., San Francisco.*

THE symptoms and physical signs which develop after an acute obstruction of the bowel are familiar to all physicians. By careful deductions it is usually possible to predicate the approximate point of the obstruction, the presence or absence of asphyxia of the bowel and, in many instances, the cause of the obstruction. Nevertheless the mortality rate, in general, remains far too high. An analysis of the records of more than one hundred patients operated upon for obstruction of the bowel at the Los Angeles General Hospital during the past year has demonstrated anew that the delay between the onset of symptoms and the appearance of unmistakable signs of grave shock and toxemia is responsible for the greater number of deaths.

FACTORS CAUSING DELAY

The factors which produce this delay are numerous, but due to their importance will be enumerated in their order of frequency:

1. Much time is often lost applying simple remedies, such as enemata, lavages and hot stupes, in the hope of relieving "gas."

2. In instances of pyloric obstruction from ulcer, faith in pills, powders, and diets not infrequently leads to unwarranted delay of operation.

3. In the more acute cases of small bowel obstruction valuable time is frequently lost awaiting the results of laboratory procedures or gastrointestinal radiographs or consultants' opinions.

4. Frequently the patient cannot be made aware of the seriousness of his condition before the progressing toxemia has made operation hazardous.

5. The abuse of sedative drugs has played a minimal part in causing delay in this series of patients.

Many of these causes of delay are beyond the control of the physician. A number, however, might have been prevented. In very few acute conditions is clinical judgment so important and laboratory studies so unimportant. A flat radiograph of the abdomen in the upright and possibly in the horizontal position, blood counts and an examination of the urine should be made at once and the results should be obtained without occasioning appreciable delay. Blood should be withdrawn for chemical analysis, but operation should not be postponed on this account. Although the results of blood chemistry determinations are quite characteristic, their importance in diagnosis in the early stages of bowel obstruction is likely to be exaggerated. Later, in the progress of the condition these results are of much value, for they give an adequate idea of the severity of the toxemia and, in addition, point to a rational therapy. Since the administration of physiological or hypertonic salt solution should be a routine procedure in all instances of suspected obstruction of the bowel the results of blood chemistry determinations should not be awaited at the expense of earlier operation.

CLINICAL SYNDROME IN BOWEL OBSTRUCTION

The exact cause of the clinical syndrome presented by patients with obstruction of the bowel is not completely known. Earlier work[1] emphasized the importance of the absorption of toxic material, either sterile or contaminated, from the bowel above the point of obstruction. This hypothetical toxic material was of unknown origin and composition, and proof of its absorption from the bowel is still lacking. It is possible that toxic material is formed in an obstructed bowel with damaged mucosa or, indeed, in a normal bowel, but it has been impossible to prove that absorption of such material through the bowel wall is frequently a cause of symptoms or disease.

Later observers have given their attention to the loss of digestive and intestinal fluid, inanition, dehydration, and the profound alterations of the physical and chemical equilibria in the body. They have also reëmphasized the differences between simple obstruction and obstruction with asphyxiated areas of bowel. In the former instance the toxemia is less severe and life may be prolonged for a greater period of time than in the latter. In dogs with simple obstruction *properly treated,* death seems to depend more on starvation than on toxemia.[2] With asphyxiated bowel, or any

* Read before the General Surgery Section of the California Medical Association at the fifty-eighth annual session, May 6-9, 1929.

Table Giving Data from Twenty-five Cases of Intestinal Obstruction

Name	Number	Age	Duration of Acute Symptoms	Amount of Vomiting	URINE			W. B. C.	Temperature	BLOOD			Point of Obstruction	REMARKS	RESULT
					Albumin in	Casts	Reaction			N. P. N.	CO_2	NaCl			
J. C.	18818	24	12 hrs.	2+	3+	0	alk	14,200	99[4]		47	429	Jejunum	Bowel viable	Cured
H. D.	44970	38	5 da.	4+	1+	0	ac	6,000	103°	33	67	231		Vomiting and diarrhea	Not operated
M. A.	27445	56	48 hrs.	4+	0	0	ac	22,000			62	346	Ilium	Cyanotic bowel not resected	Cured
Wm. W.	48704	81	12 hrs.	1+	0	0	alk		99		61	330	Ilium	Bowel not cyanotic	Cured
V. S.	45071	68	5 da.	4+	2+	0	ac	15,400	98	42	32	396	Ilium	Gangrene 6 cm. resected	Died
D. L.	47389	54	22 da.	2+	1+	0		10,100	100°		62	297	Ilium	Bowel cyanotic	Cured
F. G.	36762	38	5 da.	3+	1+	0	ac	8,600	98°			379	Ilium	Bowel good condition	Cured
A. J.	35086	40	35 da.	3+	0	0	ac	13,200	99[2]		47.1	231	Ilium	Bowel viable	Cured
M. M.	29303	28	24 hrs.	1+	0	0		8,750	98[6]	33	65	460	Ilium	Bowel viable	Cured
E. M.	28076	30	1 da.	1+	0	0	ac	18,800	99			412	Ilium	Bowel viable	Cured
A. F.	52189	69	1 da.	2+	1+	0	ac	18,100	97[2]	41	61	448	Ilium	Bowel viable	Died
R. G.	53910	59	2 da.	1+	0	0			99		60	363	Ilium	Bowel viable	Died
L. D.	27896	38	2 da.	3+	0	0	alk	7,000	98°			363	Cecum	Adhesions released	Cured
G.B.H.	47730	54	21 da.	1+	0	0	ac	29,800	99	33	58	478	Colon	Resection, partial; colon	Died
R. C.	44427	57	3 da.	2+	0	0	ac	22,000	98°	35		371	Colon	Resection partial carcinoma	Died
G. N.	496	76	2 da.	3+	1+	0	ac	15,400	98		60	462	Sigmoid	Resection sigmoid	Died
J. A. H.	2812	46	14 da.	3+	0	0	alk	11,400	98°		63	363	Pylorus	Gastro-enterostomy	Cured
W. B.	49066	40	30 da.	3+	0	0	alk	7,800	98[8]	42	65	360	Pylorus	Gastro-enterostomy	Cured
P. E.	46018	69	35 da.	3+	0	0	acid	8,500	97°	33	69	346	Pylorus	Not operated	
G. H.	37781	52	180 da.	1+	0	0	alk		98°	33	58	378	Pylorus	Gastro-enterostomy	Died
R. M.	48398	64	14 da.	3+	0	0	alk	7,200	98°	85	77	330	Pylorus	Gastro-enterostomy	Died
A. G.	5171	50	21 da.	3+	0	0	ac	10,050	99°			462	Pylorus	Gastro-enterostomy	Died
R. M.	27181	39	14 da.	3+	1+	0	ac	6,000	99[2]	63		330	Pylorus	Gastro-enterostomy	Cured
J. C.	4528	44	21 da.	3+	0	0	alk		98°		63	460	Pylorus	Gastro-enterostomy	Cured
M.		40	30 da.	3+	1+	0	alk		98°		112	600	Pylorus	Gastro-enterostomy	Died

*N.P.N. as mgm. per 100 cc. whole blood; CO_2 as vol. per cent; Cl as mgm. NaCl per 100 cc. whole blood.

other necrotic tissue free in the peritoneal cavity, grave toxemia is added to alterations produced by simple obstruction.

The use of saline solution in the treatment of obstruction of the bowel was advocated by Hartwell and Hoguet [3] in 1912. Although they believed the relief of anhydremia was the important factor their work deserves much credit. Tileston and Comfort [4] two years later proved that the nonprotein nitrogen fraction of the blood was increased in obstruction. MacCallum [5] and his associates in 1920 showed that hypochloremia and alkalosis accompanied gastric tetany produced experimentally in dogs and that the symptoms could be relieved by the administration of saline solutions. Haden and Orr [6] in 1923 published their first report, and in a series of investigations have added much to the knowledge of the alterations produced by simple obstruction.

The known facts may be briefly stated as follows: At the onset of obstruction, fluid loss occurs by vomiting and probably by secretion into the bowel lumen. This may be accompanied by the loss of as much as five grams of chlorids per liter of vomitus. As a rule the chlorid content of the blood falls rapidly. However, since the sodium content of the blood is decreased little, if at all, the resulting acid-base imbalance is partially compensated by the retention of CO_2, leading to an alkali-excess type of alkalosis. When the CO_2 volume per cent reaches about eighty-five, tetanic symptoms become manifest, but even at this time the p_H of the blood is little altered. Since sodium represents about 92 per cent of the fixed base and chlorid and CO_2 about 96 per cent of the total acid radicles of the body, alkalosis is inevitable in any chlorid loss not quickly restored. Furthermore, since "it is probable that the maintenance of a normal osmotic pressure is of more importance to life than the maintenance of a normal acid-base equilibrium," the loss of large quantities of osmotically active chlorid may be compensated by the retention of the less osmotically active nonprotein nitrogen substances in the blood although some increase in the urinary nitrogen excretion may indicate an abnormally high

body-protein metabolism. Certain alterations of minor importance also occur. The sodium and potassium content of the blood decrease but slightly. Calcium and magnesium are little changed and the quantity of sulphur and phosphorus is increased.

Complete water and electrolyte balances throughout the course of an intestinal obstruction are not available. In consequence, certain important data of great value are still lacking. The fate of the chlorids is not completely known, but observation of clinical cases makes it reasonable to assume that the loss of gastric contents by vomiting is chiefly responsible for the dehydration, increased viscosity of the blood, oliguria, and chloropenia. This explanation seems more likely when one considers that the same phenomena occur in cholera, in severe diarrhea, and in other states associated with vomiting and diarrhea. Brown, Eusterman, Hartman, and Rowntree believed that renal insufficiency might play a part in the toxemia. It seems more logical to assume that the retention of nonprotein nitrogen is compensatory to the chlorid loss. Furthermore, Blum [7] has shown that in certain types of nephritis loss of chlorids by vomiting or reductions of chlorid intake for therapeutic reasons will lead to greatly increased nonprotein nitrogen retention in the blood.

The changes encountered in intestinal obstruction: alkalosis, chloropenia, and retention of nonprotein nitrogen occur in many conditions associated with loss of gastric or intestinal juices and are not diagnostic of intestinal obstruction. Furthermore the toxemia of intestinal obstruction may be severe before these changes make their appearance and, as is well known, this is particularly true when the point of obstruction is high.

DATA FROM TWENTY-FIVE CASES

The important data from the records of twenty-five patients recently observed with various types of obstruction are summarized in the accompanying table. It will be noticed that a chloropenia was practically constant while important degrees of alkalosis or of nitrogen retention were seldom observed. The lowest blood chlorid occurred in a patient suspected at first of having an obstructed bowel but who recovered without operation. The highest chlorid content was observed in a patient with syphilis of the stomach in whom the pyloric obstruction was the indication for gastro-enterostomy. In this instance the patient vomited continuously, but the vomitus contained only traces of chlorids, yet the CO_2 content of the blood was above one hundred volume per cent for several days, and the patient was tetanic. These exceptional cases should call attention to the need of caution in formulating any hypothesis concerning the cause of toxemia and death in acute intestinal obstruction. Further studies of the acid-base balance, of the osmotic balance, and of the part played by anhydremia, inanition and toxemia may easily explain the exceptional cases encountered in any large series.

838 Pacific Mutual Building.

REFERENCES

1. Whipple, G. H., Stone, H. B., and Bernheim, B. M.: J. Exper. Med., 1913, xvii, 286.

2. Foster, W. C., and Hausler, R. W.: Arch. Int. Med., 1925, xxxvi, 31.

3. Hartwell, J. A., and Hoguet, J. P.: Jour. Am. Med. Assoc., 1912, lix, 82.

4. Tileston, W., and Comfort, C. W., Jr.: Arch. Int. Med., 1914, xiv, 620.

5. MacCallum, W. G., Lintz, J., Vermilye, H. N., Leggett, T. H., and Boas, E.: Bull. Johns Hopkins Hosp., 1920, xxxi, 1.

6. Haden, R. L., and Orr, T. G.: Bull. Johns Hopkins Hosp., 1923, xxxiv, 26.

7. Blum, L., and Weil, J.: Bull. et Mém. d. la Soc. Med. des Hôp. de Paris, 1928, xliv, third series, 1611.

DISCUSSION

Charles D. Lockwood, M. D. (65 North Madison Avenue, Pasadena).—My discussion will be limited to acute intestinal obstruction.

Little progress was made in the treatment of this condition up to five years ago, notwithstanding the great improvements in other fields of surgery. Since the epoch-making work of Orr and Haden (*Journal A. M. A.*, August 28, 1927), on blood chemistry in relation to acute intestinal obstruction, chief interest has centered in the toxemia associated with obstruction and a more rational basis has been found for treatment of this serious condition. The most fatal cases are those where obstruction occurs high up, and the toxins in the duodenum are most fatal. There has been much discussion as to the nature of these toxins, whether they are bacterial in origin, or protein bodies.

B. W. Williams of London, in the *Lancet* for April 1927, points out the importance of toxemia due to anaërobes in acute obstruction and peritonitis. The late symptoms of peritonitis are identical with those found in fatal cases of intestinal obstruction. Adynamic ileus is the result of peritonitis and the general manifestations of fatal cases of peritonitis are identical with those of the terminal stages of intestinal obstruction. The chief symptoms in common are rapid pulse, cyanosis, slight general icterus, and especially restlessness and a pathologically acute consciousness up to the very end. Williams points out (what was observed by Army surgeons) that these symptoms bear a striking resemblance to those observed in severe cases of gas gangrene. This led to investigations to determine the part played in acute obstruction by anaërobic organisms. The one most commonly found is *Bacillus welchii*. This organism is constantly present in the intestines and produces a very powerful toxin. The organism grows best in a slightly neutral medium such as is found in the duodenum. In acute obstruction and late peritonitis there is great proliferation of the *Bacillus welchii*.

Williams has been using an antitoxin prepared from this organism at St. Thomas Hospital in London for two and one-half years in acute obstruction and peritonitis associated with paralytic ileus. Only the most severe cases were treated. The series consisted of 256 consecutive and unselected cases, and there were only three deaths in the series. This is indeed a remarkable showing in a type of cases where the normal mortality is around 50 per cent.

This new knowledge together with that made available by Orr and Haden, in their experimental work on dogs, I believe has laid the foundation for a revolution in our treatment of acute intestinal obstruction. The salient facts in Orr and Haden's work are: first, the diminished chlorids in the blood; second, the increase in nonprotein nitrogen; and, third, increased CO_2 combining power of the blood. Orr also showed experimentally that a restoration of the normal chlor-

ids greatly prolonged the life of dogs with intestinal obstruction, and that jejunostomy hastened death. Dogs with intestinal obstruction live longer than those with a simple high jejunostomy. The life of dogs with high obstruction was greatly prolonged by the administration of chlorids. In view of this experimental evidence and the collected statistics in human beings, the value of jejunostomy in intestinal obstruction is of doubtful value. The mere opening of a loop of intestines in no way insures drainage. Peristalsis is essential for this process. The loops of obstructed and paralyzed bowel hang like wet rags over a rope, and only stripping of each individual loop will adequately empty the bowel of its toxic material.

This brings us to the consideration of a more thorough method of operating in early cases of obstruction. Dr. W. B. Holden of Portland, Oregon, advocates complete eventration of the acutely obstructed bowels, and the introduction of a large glass tube, secured in the bowel by a flange around which a catgut suture is tied. All obstructed loops are then quickly emptied by stripping between two fingers of the gloved and vaselined hand. He has reported a series of over one hundred cases in which the mortality has been reduced to 20 per cent in the early cases.

With all of the foregoing facts in our mind, let us outline the course to be followed by the surgeon in these acute cases of obstruction.

1. In cases diagnosed early and operated upon within twenty-four hours do not wait for blood chemistry examinations nor for x-ray findings. Open the abdomen through a lower mid-line incision and seek for the site of obstruction. If it is a band, a twist, or an intussusception that is easily relieved and the bowel is in active peristalsis, no more need be done. If there are many water-logged loops of bowel, eventrate on a hot towel, quickly empty them of their contents, restore them to the abdominal cavity and close without drainage. Either simultaneously with the operation or immediately following, give 2000 cubic centimeters of normal saline subcutaneously.

2. Cases which have been obstructed more than twenty-four hours are usually toxic. Immediate operation is often contraindicated. The surgeon is justified in taking time for a blood chemistry and restoration of the chlorids in the blood. To this end, while waiting the blood chemistry report, 500 cubic centimeters of three per cent normal salt solution should be administered by hypodermoclysis and the stomach washed out. In view of the results obtained by Williams in the use of the *Bacillus welchii* serum, if obtainable, its administration should be begun to counteract the effect of the toxins. Operation should not be performed until the chemical balance in the blood has been largely restored.

Immediate operation releasing the obstruction will only permit a lethal dose of the toxins to escape into the undamaged bowel, where it will be quickly absorbed. As soon as the blood chemistry approaches normal under the continued administration of salt solution, operation should be undertaken to remove the obstructing lesion or, if necessary, resect the gangrenous bowel. The success of operation in these advanced cases depends upon speed and accuracy, and provision should always be made for the escape of gases through a catheter introduced into the bowel above the site of obstruction in such a manner that it can be removed without reopening the wound.

The old dictum, operate immediately in acute intestinal obstruction, should no longer be followed unqualifiedly. Each case must be analyzed and the treatment adapted to the individual case. Already the beneficial effects of the new knowledge are being reflected in the mortality statistics.

❀

William J. Kerr, M. D. (University of California Medical School, San Francisco).—In the main, I am quite in accord with the views expressed by the author in this excellent paper on intestinal obstruction. There is no doubt that many lives are lost because the condition is not recognized early enough and the necessary measures taken to give relief. As the author has stated, many of these factors seem to be beyond the control of the physician. However, it appears to me that if the medical profession would join in a campaign of education the sufferers would hesitate to use home remedies where the results may be of such a serious nature. Physicians also should be more familiar with the dangers of procrastination, particularly in the case of chronic ulcer at the pylorus, and should accept the well-established rule that intestinal obstruction is one of the conditions that demands surgical intervention. We are too free in the use of cathartics and morphin in cases of vomiting, distention, or pain in the abdomen. The latter should most certainly be withheld until after a definite diagnosis is established. The time lost in waiting for results of laboratory studies in many instances endangers the life of the patient. If we would be a little more careful to analyze the symptoms and the progression and to sit down by the bedside for a careful examination of the abdomen with an analysis of the location of the distention and pain and to observe peristaltic waves, we would more often arrive at a diagnosis of the condition and determine the location of the obstruction. Quite often a plain x-ray film of the whole abdomen may reveal more correctly the site of the obstruction. Very often I have found that all the tests that are done only tend to confuse us in our decision as to treatment.

Since in most cases of acute intestinal obstruction the cause is one which requires surgical intervention and the life of the individual depends upon the correction or relief of the obstruction before toxic symptoms have become advanced, we must work with all possible haste but with the greatest clinical judgment. If a large amount of fluid has been lost by vomiting, we can assume that the chlorids are also low and no harm could come from administration of large amounts of fluids and chlorids. The question as to whether the content of the bowel should be emptied and whether the segment of the bowel should be resected depends entirely upon conditions at the time. There is no doubt that removal of large quantities of fluid from a paralyzed or inactive bowel has been of great value in treatment. I have not personally had any experience with the use of antitoxins for *Bacillus welchii*. Doctor Lockwood, in his discussion of Doctor Mason's paper, calls attention to the group of late cases where there has been much loss of fluid and where alkalosis has developed. I quite agree with him that in such cases operation is extremely hazardous and that in this very case it may be more important to the patient to try to restore the fluids and the acid-base equilibrium with a replacement of the chlorids before any operative measures are attempted.

In closing, I should like to suggest that if we are to reduce the mortality in intestinal obstruction still further, we should attempt a program of education for the medical profession as well as for the public so that this condition may be early recognized and properly treated. Great success has come from treating the diabetic through proper instruction in impending coma. Since a great majority of the acute intestinal obstructions are complications occurring in those who have had previous abdominal or intestinal conditions, we are in a position to give them certain suggestions which may save hours and, therefore, many lives when such accidents develop. Furthermore, we may prevent the use of home remedies, which are a contributing cause of mortality in many cases.

❀

Lovell Langstroth, M. D. (490 Post Street, San Francisco).—Doctor Mason reviews briefly the best modern knowledge of the physiologic disturbances caused by intestinal obstruction and reports the results of its application in twenty-five cases. I can add no further interpretation or discussion.